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The PRESIDENT enquired whether Dr. Dauber had any preference as regards the anæsthetic given. It was important to maintain anæsthesia until the application of the bandage.

Dr. BLUMFELD said that the paper seemed to him most sensible and practical, and though he did not propose to attempt any comment in general upon that very wide field covered by Dr. Dauber, he would say a few words on the important part played by anæsthetics in helping towards immediate and remote success in abdominal operations. The choice of anæsthetic was important, and chloroform should, generally speaking, be used most of the time in abdominal cases. There was no doubt that, owing to the pain and difficulty of coughing associated with the healing of a long abdominal wound, there would be an extra proclivity to bronchitis if much secretion were present in the bronchial tubes, as was likely after long ether inhalation. Another important matter was the position in which the patient was placed directly after operation. Dr. Blumfeld preferred generally to have the patient placed on the right side, the head only just raised above the level of the shoulders, and a pillow bolstering up the back and left shoulder. Some surgeons preferred in these cases to have the patient supported in almost a sitting attitude as soon as they were back in bed after operation. It was maintained that this helped to prevent vomiting, and that it was very rarely necessary to lower the patient on account of the danger of collapse. Dr. Blumfeld asked the reader of the paper for his opinion on the value of this position.

Dr. DAUBER, in reply, said that, generally speaking, he preferred to leave the choice of anæsthetics and all matters connected with their administration to the anæsthetist, on whose skill and knowledge he relied. The mind of the operator needs to be intent on the details of the operation itself. Still, he preferred chloroform where there was any bronchial or nephritic trouble. The position described by Dr. Blumfeld he considered a very suitable one.

NOTES ON THE TREATMENT OF APOPLEXY.

BY J. FOSTER PALMER, M.R.C.S., L.R.C.P.

IN apoplexy there are practically only two methods of treatment: the active and the passive. The active treatment is to trephine the skull, cut down to the seat of the brain lesion, turn out any clots of blood and effusions of serum that may be found, ligature any ruptured blood-vessels, aspirate any distended veins, and apply antiseptic astringents to diseased parts. This method, however, belongs to the future, and requires for its execution powers of diagnosis and appliances that we are not yet possessed of. It needs the aid of light rays that have not yet been invented; rays that will penetrate bone and nerve tissue, but which will be checked by blood-vessels and completely obstructed by collections of coagulated blood or effused serum. Then, too, we may be able to see if the symptoms depend, as they sometimes do, solely on abnormal distension of the veins. In this case, it might be possible, even without operation, to remove sufficient blood from the jugular to restore the balance between the venous and arterial circulation in the brain. Until we can ascertain this condition with certainty, vene-section is empirical and dangerous. As, therefore, we are not yet in possession of these desiderata, we must await the results of scientific research later in the century, and apply ourselves to the only method of treatment within our reach: the passive treatment. There is no middle method. Any intermediate system is dangerous—fatal. Treatment must be either absolutely passive or entirely active. For our present purpose, therefore, the indications are chiefly negative. I need not warn you against bleeding. It is probably never done; but less than 50 years ago it was a very prevalent and fatal practice. But there is another, somewhat similar, and, I think, equally dangerous practice, not yet entirely extinct, and that is giving aperients. It used to be a kind of routine practice, not so very long ago, to give two drops of croton oil as a matter of course. We have got out of that. But a lesser matter will serve the turn. During the last 40 years I have seen several cases of apoplexy. Of course there are cases which are evidently hopeless from the first. But there are others which, though severe, seem stationary. The worst symptoms subside. Life continues. If left alone the lesion, in many cases, will heal, and a more or less complete recovery will ensue. But if, before this takes place, an aperient be given, the patient will be dead in about two days. How it acts I cannot say. Perhaps by increasing the action of the vaso-dilator nerves; perhaps by enfeebling the nervous system generally; perhaps merely by increased muscular movement, resulting in greater blood pressure, and consequently increased effusion. I only know that aperients, however mild, are extremely dangerous, and generally, I consider, fatal. Warm injections are, I think, as bad. Possibly olive oil alone, as an enema, might be less injurious. In any case, I would leave the bowels unrelieved for a fortnight or more, *i.e.*, until the healing process was well established, rather than give an aperient, however mild. It is at this stage that such extreme caution is required. The first shock of the attack is tided over. Hæmorrhage has ceased. The brain begins to act feebly. The effused blood begins to coagulate. With the coagulation, contraction sets in, giving further relief to the compressed tissue. Arterial blood enters the vessels. Nutrition is slowly restored, and repair commences. Fibrin is deposited around the clot, forming a capsule, which becomes firmer day by day. That is, if the patient is left alone. If, at this point, an aperient is given, the process is by some means interfered with: perhaps by the withdrawal of fibrin from the blood, perhaps by mechanical movement causing too great an accession of blood, and thus breaking down the capsule, perhaps simply by diminished innervation produced from forced action of the secretory nerves and the exhaustion which follows it. Whatever the cause, the effect is fatal. We should not, at such a stage, think of forcibly exciting the motor nerves by prescribing violent muscular action by electricity or other means. We do not excite the sensory nerves by painfully irritating the entire surface of the skin. We do not excite the nerves of special sense by flashes of intense light or loud noises. Why should we excite the secretory nerves by irritating the mucous membrane of the intestinal canal? Every artificial nerve irritation is so much loss to the power of recuperation.

Blisters to the head were recommended during my recollection. I have never tried them. Their effect will probably resemble that of purgatives: forced nerve excitement, at the expense of nerve recuperative power—perhaps to a limited degree, as they affect a more limited area.

The action of ice to the head I have never been able to understand. Does it reduce the temperature of the interior of the brain through the thickness of the skull, or does it, by contracting the vessels at the surface, drive the blood somewhere else, and increase the congestion within? I pause for a reply. In either case, what is the effect on the extravasated blood or the distended vein?

Ice to the back of the neck would paralyse the vaso-constrictor nerves, and thus presumably increase cerebral hæmorrhage. Heat applied to the same part would, on the contrary, increase their action, and consequently diminish the hæmorrhage. Carried too far, it might do harm by diminishing the arterial blood supply. Still, brain rest, even with partial suspension of function, is an important item of treatment, so long as the heart and lungs are free, and the venous congestion fully relieved. There is another, a most important warning, also to be expressed in terms of negation. Never let an apoplectic patient lie on his back. This is, perhaps, the most important indication of all, and is one that is not yet sufficiently recognised. The supine position is one which, in most cases, will bring a case of apoplexy to a rapidly fatal conclusion. It is in this position that stertor is always most marked, and stertor is always an indication of danger, for apnœa follows in its wake. We have already cerebral asthenia, and we can do without this additional complication. We cannot always relieve at once the asthenia, but we can, in most cases, prevent the apnœa, and keep the case uncomplicated, and this will make all the difference between the life and death of the patient. A member of this society, Dr. Robert Bowles, has for fifty years been making observations on stertor, on animal dissections, on the dead body, on the healthy living body, as well as in pathological conditions, not only in apoplexy, but also in drowning, in anæsthetism, and other abnormal conditions. In season and out of season he has constantly insisted on the importance of this symptom in apoplexy, and I think anyone who has read his work on this subject* will be convinced of the correctness of his conclusions. I must admit that when I first saw the account of these observations, about thirty-five years ago, they did not excite much enthusiasm. I looked upon apoplexy as a fatal disease, and it did not appear to me to be a matter of very great importance whether an unconscious patient lasted four days or only three. But I have seen recoveries since then, more or less complete, and I am convinced that the exclusion of apnœa and the encouragement of blood aëration by preventing stertor are important factors in the result. It would be impossible here to go through all the experiments and demonstrations. They are set forth in the work referred to. The general principle is obvious. Stertor means, of course, some obstruction to the entry of air into the lungs. In hemiplegia we get paralysis, on one side, of the muscles of the palate. When the patient is on his back the paralysed muscles allow the tonsil to fall back and diminish the opening into the larynx. Stertor ensues, aëration in the lungs is obstructed, the supply of pure arterial blood to the brain, now more than ever necessary for the restoration of its functions, is prevented, the backward pressure causes still further distension of the cerebral veins and tissues, cerebral auto-intoxication results, and the patient dies.

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* *Stertor and Apoplexy*, by Robert L. Bowles, M.D. (Baillière, Tindall, and Co.).

If the patient is now placed on the sound side, there is sometimes little or no improvement. The paralysed muscles fall down from the affected side and diminish the size of the opening as before, though, perhaps, to a less extent, and the stertor still continues. The patient must therefore be placed on the paralysed side: then the paralysed muscles on this side will fall downwards by gravitation, while on the sound side they will be held up by muscular action, and the largest amount of air possible will be able to enter the lungs. Stertor will cease, air will enter the lungs more freely, aërated blood will be conveyed to the cerebral arteries, while the cerebral veins and tissues will be relieved of their distension.

In carrying out this treatment, there is, of course, a very serious danger to be guarded against. If the patient lies long on the paralysed side, he will almost certainly have a bedsore over the hip joint. The position must, therefore, be changed at frequent intervals by placing him for a time on the sound side, not on the back if it can possibly be avoided. The tongue falling back towards the vertebral column causes far more serious obstruction than the tongue with the muscles of the palate and uvula falling sideways. In other words, pharyngeal stertor is more obstructive than palatine. Still, after all, even a bedsore is better than suffocation if it cannot be avoided, and in thin people it comes on with extraordinary rapidity. I have had it occur once, but it healed up, and the patient got well all the same.

Stertor may be nasal, or palatine, or pharyngeal, or laryngeal. The nasal form is produced, when independent of any obstruction, by the falling of the *ala nas~~æ~~e* towards the septum from lack of muscular support when the patient lies on the sound side; this form is likewise relieved by lying on the paralysed side.* There is always, too, a tendency to breathe through the upper nostril, on account of the gravitation of blood, and consequently increased congestion of the mucous membrane in the lower nostril. This may be a further factor in the case. Stertor in the larynx, when it occurs, is generally due to œdema, or to a general relaxed condition of the parts, as hemiplegia has been shown not to affect the larynx unilaterally. Besides, relaxation or paralysis of the muscles of the larynx is not accompanied by narrowing, but by widening of the opening of the glottis. Pharyngeal and palatine stertor I have already referred to: the former occurs when the tongue falls backward in the pharynx, the latter when the paralysed muscles of the palate and uvula fall to the opposite side. In all cases, then, the line of least resistance is obtained by turning the patient, at first, on his paralysed side. When, as in some comatose cases, it is impossible to find which side is paralysed, both sides may be tried alternately, but either side will be better than the back.

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* I have tested this matter by nearly forty years' auto-obervation. I am still unable to breathe freely when lying on the right side from the lack of muscular support to the left *ala nas~~æ~~e*, the result of facial paralysis occurring thirty-eight years ago.

On looking over Bowles's cases of apoplexy, I find that most of them were fatal. They are chiefly cases of distress and discomfort relieved; of quiet, peaceful endings, of "euthanasia" I think he calls them; of slightly prolonged life, but not of recovery. It was this, I think, which at one time made me rather sceptical as to the treatment. But this need not surprise us, for he constantly gave his patients five grains of calomel, and often two drops of croton oil as well. This is enough to kill any patient, and I have no doubt many would have recovered by reason of the postural treatment if they had not been thus handicapped by something else. Indeed, the improvement in their condition shows that they were, at least, put in a state more favourable to recovery. This kind of treatment, however, is only what we should expect. Bowles's mind was so impressed with the importance of stertor and suffocation that it was diverted from other points of treatment. There are many superstitions, many dragons in the way of treatment to be attacked, and we cannot expect one man to cope with them all. This is an age of specialism, of limited possibilities. "One man, one vote." "One man, one discovery." "One man, one innovation." If a man attempts more than one, he is looked upon with suspicion. It has taken fifty years to kill this particular dragon of the supine position, and it is not dead yet; though I think now most physicians are agreed that the supine is the worst possible position for apoplectic patients. Old superstitions die hard.

This leads to a further question. If the prevention of stertor, and the free entry of air into the lungs, in order to relieve venous tension and ensure the supply of arterial blood to the brain, are all-important factors in recovery from apoplexy, it follows as a necessary corollary that in some cases the introduction of oxygen into the lungs may be advisable, and even imperative. If the size of the entrance to the lungs cannot be increased, the air that enters them may at least be concentrated by the addition of oxygen. This, of course, may be overdone. We don't want a stampede, even of arterial blood, from the lungs to the brain. The quantity of oxygen must be regulated, and probably the cessation of stertor would furnish the precise indication required.

I am sorry to say I have not tried oxygen. It will require careful watching, but there are possibilities in a moderate application of it. It is not a new remedy. It was recommended by Birch in 1868, but as he recommended it at the same time in cancer, epilepsy, scrofula, neuralgia, indigestion, and a host of other diseases, little notice was taken of it. Universal remedies always savour of empiricism. Rest for the injured brain, that the process of repair may not be interrupted. Rest for the motor nerves by keeping the muscles quiet. Rest for the nerves supplying the heart and lungs by keeping up the access of air and preventing the accumulation of venous blood. Rest for those of digestion and secretion by light, by easily assimilable food, and the avoidance of mucous excitants. Rest, too, for the nerves of sensation and special sense. Times may change. New methods may arise. But, *at present*, this is practically the summing up of our knowledge and experience of the conditions most favourable to recovery from this dangerous cerebral lesion.

Prevention does not come strictly within the scope of the title of this paper. Patients, as a rule, do not ask our advice on the subject. They prefer to live their own lives, to overload their digestive organs, to dissipate their nervous power, and auto-intoxicate their blood and tissues till stopped by actual disease. Or perhaps. short of this consummation, they find their powers diminishing, their appetite failing, and resort to patent medicines, tonics, "pick-me-ups," what not, with the usual result of shortening the already shortened span of life.

Meanwhile, if they do ask our advice, we can sometimes give them warnings which will, at least, prevent their running into unnecessary pitfalls. It has been said that a man is as old as his arteries. That is, the length of his life depends, *ceteris paribus*, on the continued elasticity of these vessels. If calcification takes place in their middle coats, they are converted into brittle, inelastic tubes, and will give way on slight provocation. If the blood contains the slightest excess of calcium, it will sooner or later be deposited in some of the tissues. It may, no doubt, sometimes find a resting place in stones in the gall bladder, the urinary bladder, or the pelvis of the kidney. Perhaps it is fortunate if it does. But it is more likely to find its way to the arteries. It may be said that it will not form a deposit unless there is some inflammatory condition of the internal coat. But this may occur in a local and limited form at any time and in any place, and it is obvious that the more calcium the blood contains, the more chance there will be of its being deposited. In middle life and after it is not required by the bones. They are full long ago, and the arteries will form a convenient receptacle for it, and its mere presence in the blood will often be sufficient to bring about the preliminary irritation. Calcium *does* exist in the blood in solution, its solution being favoured by the presence of free carbonic acid.

It is important, therefore, for adults, at any rate for all above 35 or 40, to avoid lime salts as far as possible. Water, of course, contains them, especially when "hard," as it is called; in which case it is better boiled: most people will prefer it in the form of tea, or beer, or coffee, as boiled water is rather tasteless. But far more injurious in this respect is the habitual taking of mineral waters, most of which contain a considerable quantity of lime. These are especially insidious, because they often contain free carbonic acid as well, which renders the calcium salts more soluble in the blood. But worst of all in this respect are those patent medicines which contain large quantities of lime salts, such as Parrish's food, Easton's syrup, Fellows's hypophosphites, glycero-phosphates, Bynin Amara, Scott's emulsion, Angier's emulsion, *et hoc omne genus*, which, however good they may be for children, will tend to shorten life if taken by those at all beyond middle-age, both by increasing vascular tension and by promoting the tendency to calcification of the vessels.

The PRESIDENT mentioned that Professor Clifford Allbutt had pointed out that the rise of blood pressure following apoplexy probably counteracted the squeezing out of blood from the cardio-respiratory centres by the increased intra-cranial pressure, and therefore it should not be lowered by venesection. As regards withholding aperients and enemata, it was a question whether the straining and disturbance which follow prolonged constipation might not be more harmful than an early, although perhaps not immediate, attention to this function.

Mr. HAROLD BARWELL said—Dr. Foster-Palmer has alluded to the stertor of apoplexy. In my opinion, this is due not so much to the paralysis as to the flaccidity of the muscles of the palate and tongue, so that when the patient lies on the back the tongue, and with it the epiglottis, drops backwards over the laryngeal aperture. The noise is stertor, not stridor, such as occurs in some forms of laryngeal paralysis, for the vocal cords are practically never paralysed in hemiplegia. The immunity of certain muscles is one of the most interesting points in the study of paralysis resulting from apoplexy. It will be found that two features are characteristic of those muscles which escape:—(1) they are muscles which habitually act in association with their fellow of the opposite side, and (2) they are muscles which perform primitive and vital actions, low down in the scale of evolution. In the former case they escape because they are controlled from both cerebral hemispheres; and with regard to the latter, just as in lower animals like the frog the cerebral hemispheres are of little importance, so in man the ancient vital functions are strongly represented in the bulbar nuclei, while the cerebral centres are comparatively insignificant. A good instance of this is furnished by facial paralysis: in a lesion of the nerve itself all the muscles it supplies are motionless, but when the injury is above the bulbar nucleus, or in the cortex, the frontalis and orbicularis palpebrarum are usually spared, for these muscles habitually act bilaterally. And it sometimes happens that the muscles of the lips, which cannot be moved voluntarily, nevertheless move quite well in response to the emotions, as in smiling; showing that a primitive reflex movement is conserved, while the later voluntary action is destroyed. The vocal cords, then, escape for both these reasons. The larynx subserves two functions, the vital primitive and ancient function of respiration, and the more modern one of phonation; the abductor muscles act for the former and the adductors for the latter function. The cortical representation of the abductor movement is very insignificant—you cannot voluntarily abduct your vocal cords—and the respiratory excursion of the cords is carried on normally in dogs after ablation of both cerebral hemispheres; it follows that no cortical lesion will produce abductor paralysis. There is, however, a well marked centre for adduction on both sides of the brain, but it is found that stimulation of either centre causes adduction of both ends, and consequently an unilateral lesion of the cortex will cause no paralysis, while destruction of both centres or their efferent fibres will produce adductor paralysis of both vocal cords. Such a bilateral lesion as would cause adductor paralysis is, of course, extremely rare, but a few cases have been recorded, notably one by Eisenlohr, in which laryngoscopic examination showed bilateral adductor paralysis, and *post-mortem* partial degeneration of the posterior limb of the internal capsule was found on both sides, thus confirming in a remarkable way the experimental conclusions. However, the glottis is widely open in adductor paralysis, so that it may be safely said that the stertor of apoplexy is never in any way due to paralysis of the vocal cords.

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